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INTRACRANIAL ARTERIO-VENOUS ANEURISM OR PULSATING EXOPHTHALMOS

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SINCE 1809, when the phenomena of pulsating exophthalmos was first described, until June of 1923, there have been 588 instances of this malady recorded in the literature. This includes the 106 cases up to 1880 collected by Sattler,[1] the 138 cases of the combined series of Keller,[2] 1898, and Reuchlin,[3] 1902, the 69 of de Schweinitz and Holloway,[4] 1907, the 88 cases of the combined series of Bedell,[5] 1915, Rhodes,[6] 1916, Zentmeyer,[7] 1916, and Von Nagy,[8] 1919, and the 131 cases collected by the author.

The purpose of this paper is to gain information concerning the etiology and pathology of pulsating exophthalmos by the analyses of the entire series and especially to study the question of treatment, for thus far the therapeutic results have been quite unsatisfactory. An attempt will also be made to explain the various clinical phenomena associated with this disorder and three cases of the author's own experience will be reported in some detail.

HISTORICAL

Pulsating exophthalmos was first described in 1809 by Benjamin Travers,[9] demonstrator of Anatomy at Guy's Hospital. He had no post-mortem evidence on which to base his conclusions, but believed the condition was confined entirely to the eye and its etiology was that of "Aneurism by Anastomosis" or cirsoid aneurism of the orbit. Travers found that compression of the common carotid artery of his patient caused the bruit to stop and the exophthalmos to decrease and he therefore instituted the surgical treatment of ligation of the common carotid. Thus he was not only the first to describe the condition, but was the first to describe its surgical treatment even in the days before the discovery of anæsthesia. (See Fig. 1, a reproduction of engraving from Travers' original article).

Three years later, in 1812, Dalyrample[10] reported the second case of pulsating exophthalmos and accepted Travers' idea concerning its etiology, and as did the subsequent writers up to 1823. However, in this year, Guthrie[11] performed the first autopsy on one of these cases and instead of finding a cirsoid aneurism as had been supposed, he found a nut-size aneurism of the ophthalmic artery and thus he advocated this as the etiology of all previously reported cases of pulsating exophthalmos. In 1837, Warren[12] of Boston reported the first case of pulsating exophthalmos following trauma.

When in 1839, Busk[13] confirmed Guthrie's findings by autopsy of another case, it soon became accepted, at least in England, that aneurism of the ophthalmic artery was the cause of this clinical complex. However, in France in 1835, even four years before

Busk's report, Baron[14] purposed a new etiology, being the first to discover at autopsy a communication between the cavernous sinus and the internal carotid, and thus established the fact that the cause of pulsating exophthalmos might be an intracranial rather than an extracranial disorder. So brief was his report, however, that it escaped general notice, yet he should have the credit of establishing the most important point in the etiology of pulsating exophthalmos. In 1841, Gendrin[15] reported an autopsy upon a case of pulsating exophthalmos and found as did Baron a communication between the internal carotid and cavernous sinus. Nélaton,[16] in 1856, found another such communication at autopsy, and in 1857, the following year, Hirschfeld,[17] also in France by another autopsy report confirmed the idea of an arterio-venous communication.

FIG. 1.—Engraving of Travers' patient before and after operation, from Med. Chir. Trans. 1813, vol. ii. This was the first description of pulsating exophthalmos.

Brainard,[18] Professor of Surgery at Rush Medical School, in 1851, cured a case of pulsating exophthalmos by injection of a coagulating fluid into the dilated veins about the orbit, yet the patient lost the vision in that eye. In 1856, digital compression was first recommended and used successfully by Professor Gioppi,[19] of Padua, Italy, for treatment of a case of spontaneous pulsating exophthalmos, and in 1857, bilateral ligation of the carotid was used by Buck,[20] of New York.

In 1870, Delens[21] of Paris, published an exhaustive monograph on arterio-venous communication between the cavernous sinus and the internal carotid. Although French writers after 1835 recognized the intracranial origin of most cases of pulsating exophthalmos, the English continued to attempt to explain the condition on a purely orbital basis. Thus in 1854, Curling,[22] of London, supported the view of Busk concerning the etiology and in 1858, Bowman and Hulke,[23] of London, recorded a case with autopsy in which they found a dilated ophthalmic vein but no intracranial lesion.

In 1859, Nunneley,[24] Chief Surgeon of Leeds Eye and Ear Infirmary, thought the etiology was a false traumatic aneurism of the eye, or less often an aneurism of the ophthalmic artery. Nunneley's next paper in 1864, brought the first admission by English authors that the condition might be of intracranial origin. He gave up his former idea of false aneurism and believed that an obstruction to the return flow from the eye through the ophthalmic vein to the cavernous sinus was to be held accountable.

This obstruction he suggested might be a collection of serum, fibrin, pus, or a tumor. The spontaneous cases he thought were usually aneurisms of the internal carotid or of the ophthalmic artery immediately after its origin.

In 1853, the first case reported which subsided spontaneously without digital compression or ligation was described by France[25] of Guy's Hospital and in 1874, Lansdown,[26] Surgeon at Bristol General Hospital, cured a traumatic case by ligation of the varicose vessels at the inner canthus of the eye, this being the first recorded case cured by an orbital operation.

In 1875, Rivington's,[27] London, notable paper on "Pulsating Tumor of the Orbit"

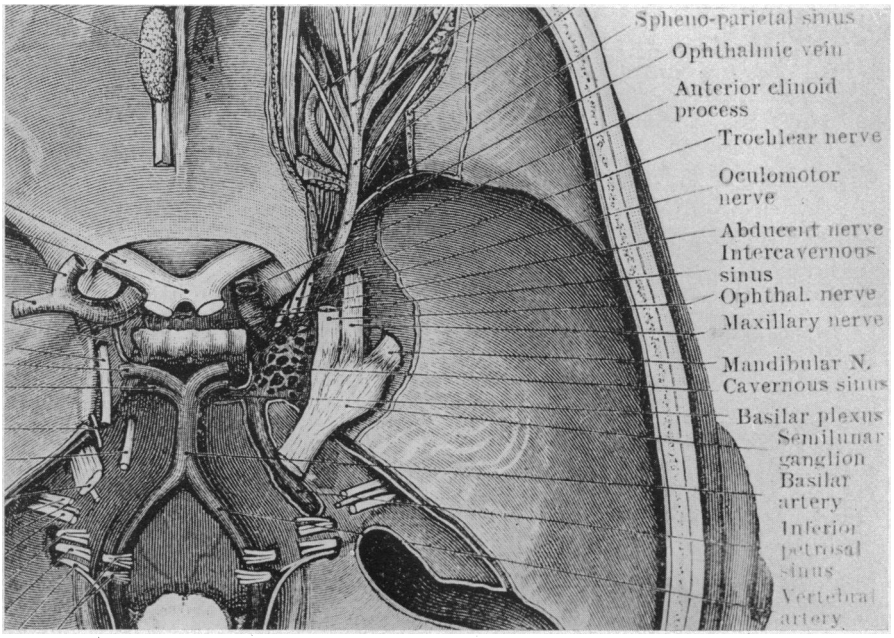

FIG. 2.—The base of the cranium with dura mater undisturbed. Note the relation of the cavernous sinus with the internal carotid artery and with the 2nd, 3rd, 4th 5th and 6th cranial nerves. (From Cunningham's Anatomy, Wm. Wood & Co.)

appeared and was the most comprehensive work on the subject up to that time. Rivington recognized arterio-venous communication as the etiology of most cases, but also mentions aneurism of the ophthalmic artery and morbid conditions of the orbital veins and intracranial sinuses.

Sattler,[1] Berlin, in 1880 collected 106 cases which was a complete list of the reports in the literature up to this date. He too believed that the etiology of most cases was an arterio-venous communication and that in previously reported autopsies this communication had been frequently overlooked.

Similarly, Keller,[2] in 1898, and Reuchlin,[3] in 1902, discussed in inaugural dissertations in Germany the etiology of pulsating exophthalmos and collected additional cases from the literature.

Murray,[28] New York, in 1904 first ligated the internal carotid in treating pulsating exophthalmos and had successful results.

The next large study of the subject was made by de Schweinitz and Holloway,[4] Philadelphia, in 1907. They assembled information from all previously reported cases, making a total of 313 and their figures showed that although a communication between the internal carotid and cavernous sinus was the most frequent cause, yet a certain

number of cases were due to an aneurism of the ophthalmic or the internal carotid artery, or to tumor of the orbit.

Since this most memorable monograph by de Schweinitz and Holloway numerous

TABLE I.

Showing Proportion of Males and Females.

Etiology	Number of cases collected	Number of males	Per cent. males	Number of females	Per cent. females	Sex not stated
Spontaneous						
Author's series (1923)	38	6	18.18	27	81.82	5
Combined series of Bedell (1915); Rhodes (1916); Zentmeyer (1916); and Von Nagy (1919)	17	6	37.5	10	62.5	1
De Schweinitz and Holloway series (1907)	14	6	42.85	8	57.14	0
Combination of all series preceding 1907 including Rivington's, Sattler's, Keller's, Reuchlin's, etc., series	57	12	23.64	41	77.35	4
Total spontaneous cases	126	30	25.87	86	74.13	10
Traumatic						
Author's series	83	60	84.51	11	15.49	12
Combined series of Bedell (1915); Rhodes (1916); Zentmeyer (1916); and Von Nagy (1919)	67	56	86.15	9	13.84	2
De Schweinitz and Holloway series (1907)	54	31	65.95	16	34.04	7
Combination of all series preceding 1907	214	147	73.5	53	26.5	14
Total traumatic cases	418	294	76.76	89	23.24	35
Etiology Not Stated						
Author's series	10	6		0		4
Combined series of Bedell (1915); Rhodes (1916); Zentmeyer (1916) and Von Nagy (1919)	4	1		0		3
De Schweinitz and Holloway series (1907)	1	0		0		1
Combination of all series preceding 1907	29	0		0		29
Total	44	7		0		37
Grand total all cases	588	331	65.42	175	34.58	82

case reports have appeared, yet no complete assembly of them has been made and hence no conclusions have been drawn.

Etiology.— (See Table I.) Of the entire series of 588 cases, there have

been 544 cases in which the etiology was stated. Of these, 126, or 23.16 per cent., were spontaneous and 418, or 76.84 per cent., were traumatic in origin. In the series of cases collected by the author the percentage of spontaneous origin was somewhat higher for out of 121 cases in which the etiology was stated, 38, or 31.40 per cent., were spontaneous, and 83, or 68.60 per cent., were traumatic in origin. By reference to Table I, it will be seen that in the spontaneous type the female predominates and in the traumatic type the

TABLE II.

Age in Pulsating Exophthalmos.

Ages	Author's series	Combined series of Bedell, Rhodes, Zentmeyer and Von Nagy	Series of De Schweinitz and Holloway	Total
Cases of Spontaneous Origin.				
1–10	1	0	1	2
11–20	2	2	0	4
21–30	4	3	3	10
31–40	4	0	4	8
41–50	4	3	0	7
51–60	8	2	2	12
61–70	3	1	1	5
71–80	5	3	1	9
81–90	0	1	0	1
Age not stated	7	2	2	11
Total.....	38	17	14	69
Cases of Traumatic Origin				
1–10	0	0	7	7
11–20	18	6	5	29
21–30	16	13	12	41
31–40	13	12	5	30
41–50	9	9	9	27
51–60	5	3	6	14
61–70	1	1	0	2
71–80	0	0	0	0
81–90	0	0	0	0
Age not stated	21	23	10	54
Total.....	83	67	54	204

male predominates. Thus, of the former type we find 74.13 per cent. women and of the latter type 76.76 per cent. men. Of the author's series 81.82 per cent. of the spontaneous cases occurred in women and 84.51 per cent. of the traumatic cases were in men.

As may be seen from Table II, the average age of the spontaneous type is greater than that of the traumatic type. Thus the average age of the spontaneous cases included in this table is near the end of the fifth decade while the average age of the traumatic cases is near the end of the third decade. The average ages in the author's series were forty-eight years for the spontaneous type and thirty-two years for the traumatic.

The left eye is involved more often in the spontaneous type while the right eye is slightly more frequently involved in the traumatic type. The

TABLE III.

Showing Proportion of Involvement of Right or Left Eye.

Etiology	Involving	Author's series	Combined series of Bedell, Rhodes, Zentmeyer and Von Nagy	De Schweinitz and Holloway series	Combined Sattler, Reuchlin and Keller	Totals	Per-centages
Spontaneous..	Right Eye	8	8	4	22	42	33.33
	Left Eye	18	4	6	30	58	46.03
	Both Eyes	6	2	2	2	12	9.53
	Not Stated	6	3	2	3	14	11.11
	Total	38	17	14	57	126	
Traumatic....	Right Eye	28	30	25	82	165	39.47
	Left Eye	25	24	21	87	157	37.56
	Both Eyes	14	8	5	31	58	13.88
	Not Stated	16	5	3	14	38	9.09
	Total	83	67	54	214	418	
Etiology not Stated.....	Right Eye	0	1	1	0	2	
	Left Eye	0	0	0	0	–	
	Both Eyes	0	0	0	0	–	
	Not Stated	10	3	0	29	42	
	Total	10	4	1	29	44	

TABLE IV.

Table of Autopsy Finding in Pulsating Exophthalmos.

Lesion	Spontaneous		Traumatic		Total
	Autopsies collected by author	All previous reported autopsies *	Autopsies collected by author	All previous reported autopsies *	
Arterio-venous communication.....	3	7	4	10	24
Thrombosis of cav. sinus and ophth. vein with probable art.-ven. communication.....	0	6	0	2	8
Aneurism of int. carotid.....	1	2	0	1	4
Aneurism of ophth. artery					
Within orbit.....	0	2	0	0	
Outside orbit.....	0	1			3
Tumors of orbit.....	1	6	0	0	7
Lesion not discovered.....	1‡	3†	0	0	4
Total.....	6	27	4	13	50

* Includes 19 autopsies collected by Sattler; 9 by Keller; 2 by Reuchlin; and 11 by De Schweinitz and Holloway.

† Two of these 3 were patients in whom the pulsating exophthalmos had been cured and patients died of other cause.

‡ Incomplete autopsy.

figures may be seen in Table III. A bilateral pulsating exophthalmos occurs more frequently in the traumatic group than in the spontaneous group.

Pathology.—Up to 1907 (de Schweinitz and Holloway), there were 40 autopsies upon cases of pulsating exophthalmos recorded in the literature. Since that time the author has been able to add 10 to this number, making a total of 50 post-mortem examinations. See Table IV. Of this number, 33 were performed upon spontaneous and 17 upon traumatic cases. Of the 33 autopsies on spontaneous cases there were 4 in which no lesion was found. In two of these, however, the pulsating exophthalmos had been cured some time previous to death and in a third case only an incomplete examination was made. Of the 30 remaining cases there were 16, or 53.33 per cent. in which communication or a probable communication between the internal carotid and cavernous sinus was found, 7, or 23.33 per cent. in which were tumors, 3, or 10 per cent. aneurisms of the internal carotid, 3, or 10 per cent. in which aneurism of the ophthalmic artery was revealed, and 1, or 3.34 per cent. in which no lesion was found. The findings of 17 autopsies upon traumatic cases is quite a different story, for 16, or 94.12 per cent. of these proved to be arterio-venous communication of the internal carotid and cavernous sinus, and only one case as aneurism of the internal carotid.

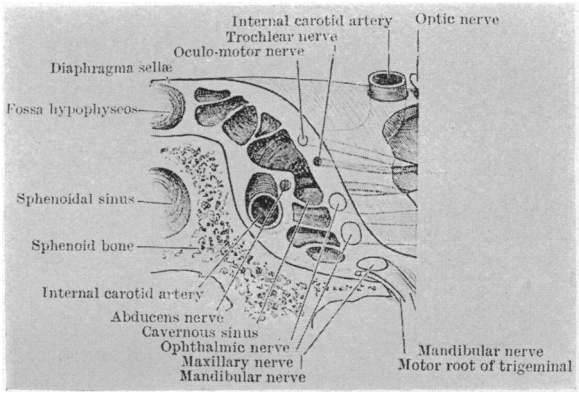

FIG. 3.—Cross-section showing internal carotid within the cavernous sinus and the proximity of the 3rd, 4th, 5th and 6th cranial nerves. (From Cunningham's Anatomy, Wm. Wood & Co., Publishers, 51-5th Avenue, New York, N. Y.)

Although arteriosclerosis has been a prominent feature in many of the spontaneous type there are very few cases in which the history suggests lues. There have been, since the time of the Bordet-Wassermann reaction, 19 cases on which this test was performed. Of this number 2 were positive and 17 negative. One of the cases with a positive reaction was a thirteen year old lad having a traumatic type and the other was a woman of seventy-eight years with a spontaneous type in which autopsy showed a tumor of the orbit. From the evidence available we would therefore conclude that lues is not a contributing factor.

In summarizing the pathology it may be stated from the scant information at hand that practically all cases of the traumatic type of pulsating exophthalmos are due to an intracranial arterio-venous communication. Of the spontaneous type only a little over one-half of the cases can be attributed to this cause, while about one-quarter are caused by tumor and another quarter by simple aneurism of either the internal carotid or ophthalmic artery.

Anatomical Considerations.—The anatomical boundaries of the cavernous sinus and the close relation to the internal carotid are clearly shown in Fig. 2 from Cunningham's Anatomy. The proximity of the 2nd, 3rd, 4th, and 6th cranial nerves, and ophthalmic division of the 5th cranial nerve to the cavernous sinus may be readily seen. A cross-section of the cavernous sinus, Fig. 3 from Grey's Anatomy, shows the internal carotid artery

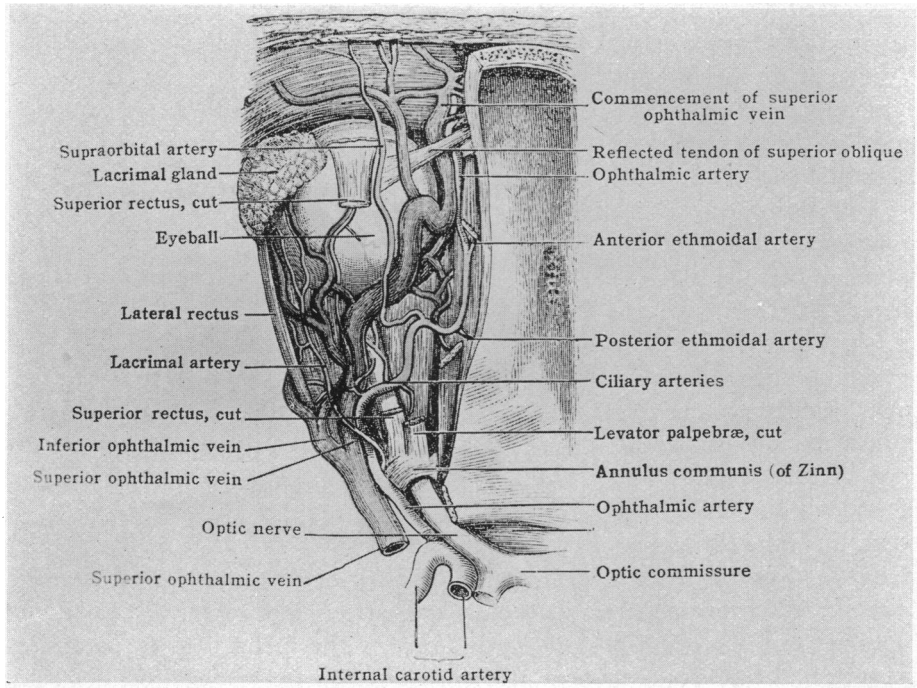

FIG. 4.—The left ophthalmic artery and vein. Note the venous plexus behind the orbit. Exophthalmos associated with intracranial arterio-venous fistula of the internal carotid and cavernous sinus may be caused by the dilatation of these pulsating vessels pushing the eye forward. (Morris' Anatomy, P. Blakiston's Son & Co.)

lying within the sinus with the 3rd, 4th, 6th and the ophthalmic and maxillary divisions of the 5th cranial nerve.

Rawlings[29] has found that 70 per cent. of the fractures of the base of the skull involve the body of the sphenoid bone. Both internal carotid artery and cavernous sinus are comparatively immovable in this region so that it is quite natural that an underlying fracture might rupture or injure their adjacent walls. The cases of actual rupture are those clinically in which the patients hear the bruit immediately upon return of consciousness, while those who hear the bruit only some days or weeks after the accident are cases in which only a damage of the vessel walls has occurred, which later ruptures. A penetrating wound, too, may have such a course that it comes in contact intracranially with both internal carotid artery and cavernous sinus. It thus may rupture or weaken the adjoining walls of these two vessels.

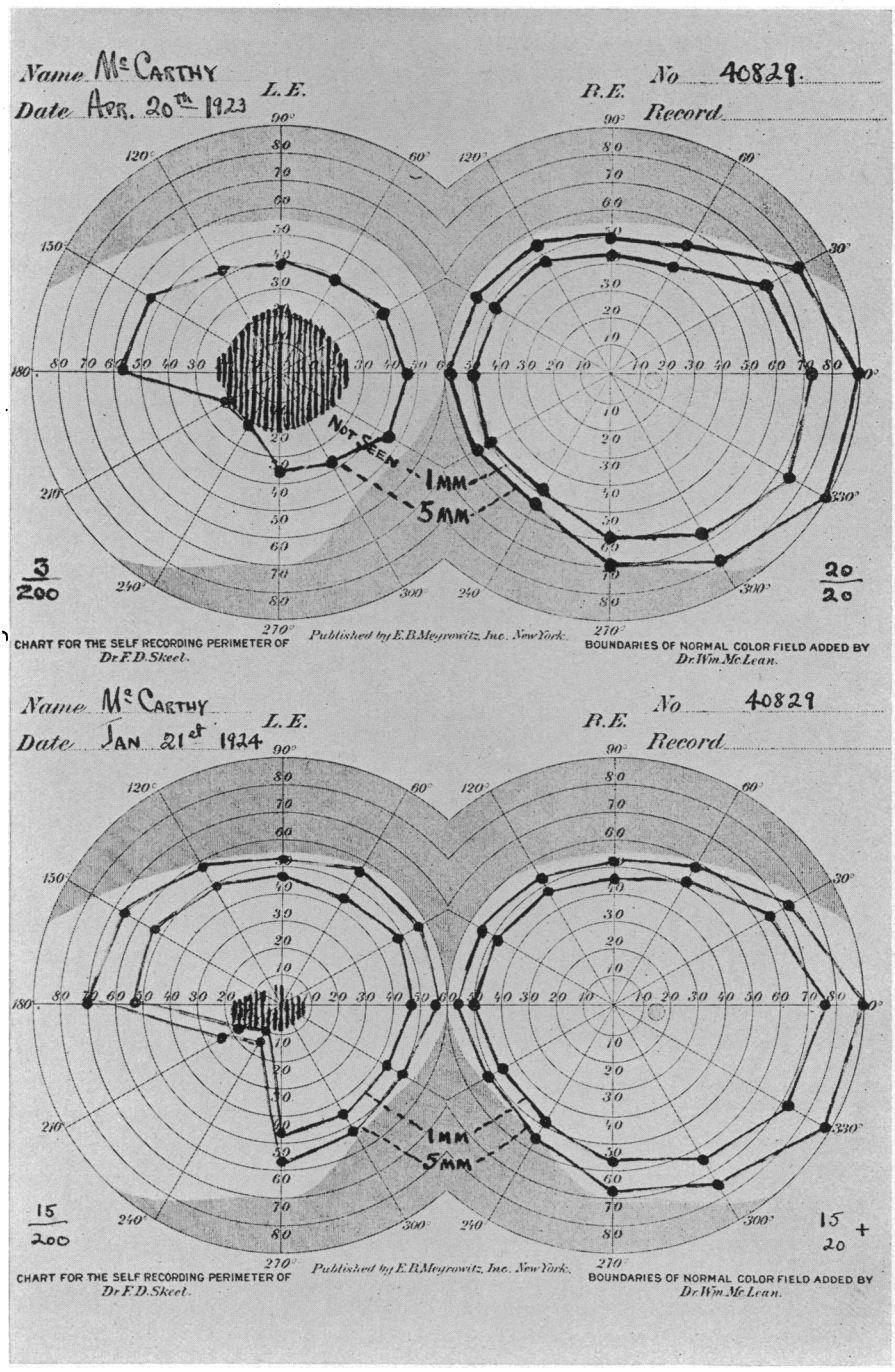

FIG. 5.—Chart of visual fields of Case I before and after operation. Note the decrease in size of the central scotoma. (By courtesy of E. B. Meyerowitz Surgical Instrument Co.)

The mechanism of production of a pulsating exophthalmos spontaneously without an injury is more difficult to explain. This may be due to a diseased and weakened condition of the walls of the adjoining vessels. Again, it seems probable that an arterio-venous communication may occur by a rupture of a simple aneurism in the portion of the internal carotid which lies within the cavernous sinus or in its immediate neighborhood. The spontaneous type of pulsating exophthalmos, of course, may be due also to a simple aneurism of the internal carotid or ophthalmic artery, or even to tumor of the orbit.

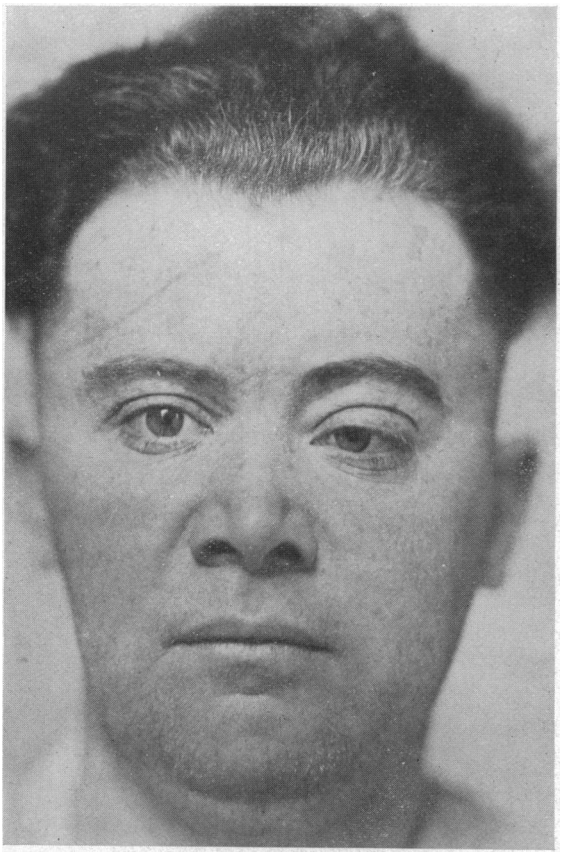

FIG. 6.—Case I before operation. Note the exophthalmos and internal strabismus of left eye and the ptosis of left upper lid infringing on pupil.

The enormously dilated and pulsating ophthalmic vein lying behind the orbit affords at least one explanation of the exophthalmos and orbital pulsation associated with the phenomena of arterio-venous communication between the internal carotid and cavernous sinus. Figure 4 of the orbital veins after Poirier and Charpy show the plexus behind the orbit, which takes part in this dilatation.

The explanation of the cause of the very large, pulsating, vascular masses above the internal angle of the eye is a question which naturally arises. One would suspect with a fistula between the internal carotid and the cavernous sinus that the rush of arterial blood would be amply taken care of in the great intracranial venous sinuses. This, however, is not true, for in the majority of cases much of the arterial blood finds its way into the superior ophthalmic vein forming large dilated swellings. The anatomical explanation of this fact, I think, is that the ophthalmic veins have no dense tissues surrounding them such as the bone and dura which surround the superior and inferior petrosal sinus, the other exists of the cavernous sinus.

The various cranial nerves may be involved in four very different ways by four very different types of mechanism: First, that of direct injury of the nerve by the fracture or contusion which causes the rupture of the cavernous sinus and internal carotid. Thus the 1st, 2nd, 3rd, 4th, 5th, and 6th

cranial nerves are often taken and less frequently the 7th and 8th. These signs are present soon after injury and perhaps are augmented by slow hemorrhage. The visual fields of Case I shown in Fig. 5 are an example of this type of injury. Secondly, cranial nerve signs may be caused by pressure from actual dilatation and hypertrophy of the vessels on either side of the arterio-venous communication. The cranial nerves affected by this mechanism are the 2nd, 3rd, 4th, 6th and the ophthalmic division of the 5th, and the signs usually appear some days or weeks after the injury. The effect of a third type of mechanism is limited to but one cranial nerve. It is due to pressure upon the supraorbital nerve near its exit from the supraorbital foramen by the enormous pulsating dilatations of the superior ophthalmic vein and its branches. Thus in the author's second case it seemed probable that this was the mechanism for the slightest touch with the finger in the region of the supraorbital foramen would often cause sharp and uncomfortable paræsthesias over the left frontal region. The fourth mechanism is limited to the 2nd nerve and is of purely circulatory nature. Thus it seems that the short circuiting of arterial supply or even venous congestion may cause optic trophy.

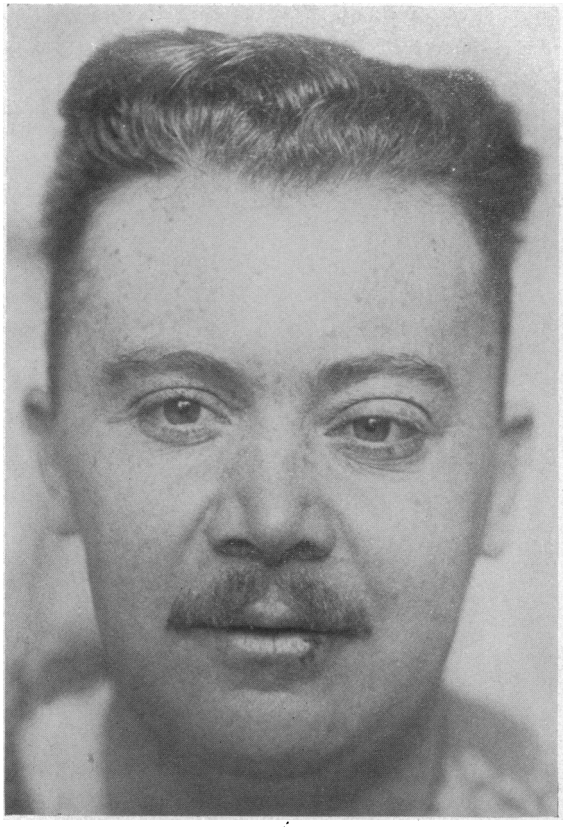

FIG. 7.—Case I two weeks following ligation of left internal carotid. Note improvement in the exophthalmos, the ptosis and the internal strabismus.

Symptoms and Signs.—The following case reports illustrate the subjective and objective findings in this rare condition of pulsating exophthalmos. Cases I and II are from the division of neurosurgery of the University of California Hospital, Case III a San Francisco Hospital patient which the former Resident Surgeon, Dr. Ray Kistler, was good enough to send to us for examination. A fourth case will be merely mentioned which was a patient at the Peter Bent Brigham Hospital on Doctor Cushing's wards, that came under my care during my service as Neurosurgical Resident.

CASE I.—*Accident. Resulting Pulsating Exophthalmos. Digital Compression of Carotid No Improvement. Ligation of Internal Carotid. Marked Improvement.*

University of California Hospital No. 18043, an American male, aet, twenty-five, an electrician by occupation had a negative family history except that father died aet, forty-six of apoplexy. Past history was negative. Present illness: On September 25, 1922, the patient was in an automobile accident and was unconscious for forty-eight hours, with profuse bleeding from the right ear, nose and mouth. There was no subsequent memory of the next five days, but on fully regaining his senses, he noticed numbness of the left side of the face, stopping at the midline, deafness of right ear, and with the left eye his vision was only sufficient to distinguish between light and darkness. Six weeks after accident, the patient was first aware of a blowing bruit within his head, louder on the left side, which he described as sounding like "the exhaust of a steam valve". At this time the vision in the left eye was still defective and soon an internal strabismus and exophthalmos of O.S. appeared. He then first noticed a bilateral loss of smell. On January 4, 1922, the patient entered University of California Hospital. At this time physical and neurological examination showed a well developed, very muscular young man with a left-sided pulsating exophthalmos (see Fig. 6), a systolic blowing bruit best heard over left eye and left temporal region, and an external rectus palsy of O. S. with internal strabismus. There was some engorgement of the small veins at the inner canthus and of the upper lid, but without distinct pulsation of them. The left pupil reacted sluggishly and there was marked ptosis on this side. Although definite subjective symptoms of numbness over the 1st and 2nd divisions of the left trigeminal were present, there were no objective signs. Hearing was diminished in the right ear, there was a bilateral loss of olfactory sense and visual acuity of O. S. was such that patient could see only the 20/100 letters when holding an ordinary acuity chart in his hand; acuity of O. D. was 20/20. Ophthalmoscopic examination of O. S. showed a pale disc with slightly hazy outline and dilated tortuous veins; O. D. normal. Visual fields showed defect (see Fig. 5), an X-ray of skull showed a left frontal fracture. Blood-pressure was 128/95. Compression of the left common carotid stopped the bruit subjectively and objectively while compression of the temporal or facial arteries or the carotid on the opposite side had no effect. Blood Wassermann and urine were negative. *Treatment:* For about three weeks the patient was kept quiet in the hospital and digital compression applied. Due to the heavy musculature of his neck, compression could not be maintained for over 10 to 15 minutes at a time. Even a slight movement of the sterno-mastoid muscle or the act of swallowing would cause the artery to slip away from the compressing finger. The patient was then sent home where the same treatment was continued for

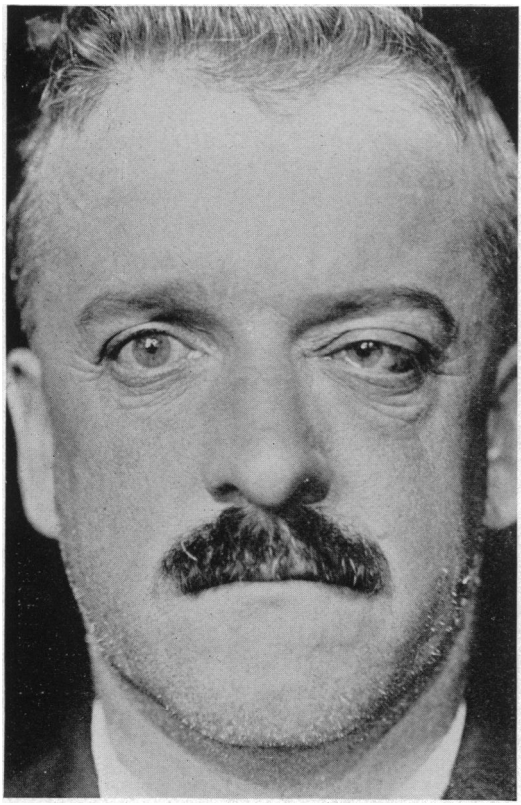

FIG. 8.—Case II before operation. Note exophthalmos, internal strabismus and ptosis.

eleven weeks without improvement. On May 1, under local anæsthesia the left internal carotid was exposed and compressed with a Crile clamp for thirty minutes. During this time no weakness, nor numbness of the opposite extremity developed, so a permanent double ligature was made. This shut off the bruit subjectively, but objectively it was still present, yet diminished to about one-tenth of its previous intensity and was of a very much higher pitch. During the following weeks the bruit became even more feeble and often could not be heard at all. It could be entirely shut off by compression of the opposite carotid. The exophthalmos improved remarkably, the internal strabismus largely disappeared and ptosis became less marked (see Fig. 7). The patient went back to work July 1, 1923, and reported that he had been working steadily when seen again on January 21, 1924. At this date his appearance was the same as that shown in Fig. 7. Visual fields showed same defect in O. S. as that noted before operation. The central scotoma, however, had decreased in size and usual acuity was somewhat improved (see Fig. 5). Nevertheless, examination of the fundus O. S. showed increased pallor of the disc with increased distinctness of the cribiform markings. The blood-vessels appear normal. A faint bruit, barely audible, was heard over the left orbit and in the left temporal region. This is, however, not heard by the patient except occasionally at night. The defect in olfactory sense was still evident although not as well marked as previously. Auditory acuity on the affected side, the right side, was almost normal.

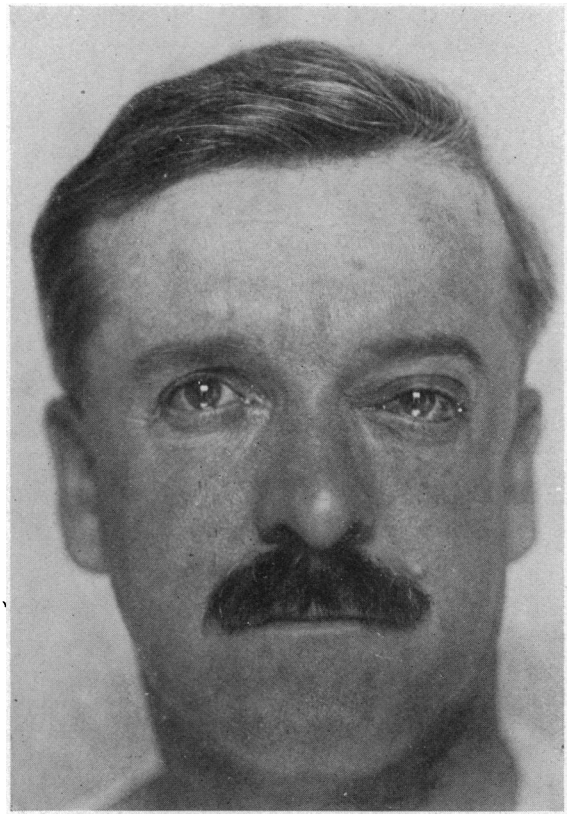

FIG. 9.—Case II two weeks after ligation of left internal carotid. Note improvement in exophthalmos, strabismus and ptosis.

CASE II.—*Accident. Pulsating Exophthalmos. Digital Compression of Carotid. No Improvement. Ligation of Internal Carotid. Improvement. Ligation of Superior Ophthalmic Vein. Further Improvement.* University of California, Hospital No. 19647-A an Irish born American male, aet, forty-two, a drygoods clerk by occupation, had an insignificant family history. Past history was negative except for malaria aet thirty-five; Neisser aet eighteen; and excessive use of alcohol and tobacco. Present illness: On September 25, 1922, while intoxicated, was struck by a motor truck, was unconscious for one and one-half hours and had considerable bleeding from his nose, although for the next forty-eight hours the patient seemed conscious, he had no subsequent memory of this time. He first remembered severe left frontal and partial headaches and a blowing noise within his head. Five days after accident there was diplopia and the left eye turned inward but exophthalmos was not noted until about five weeks later. About four months after accident he first was aware of paræsthesias over ophthalmic division of the left trigeminal

nerve and he entered the University Hospital, February 6, 1923, almost four and one-half months after injury. At this date physical examination and neurological examination showed a well marked pulsating exophthalmos O. S. (see Fig. 8), a systolic bruit heard loudest over O. S.; marked dilatation of angular and naso-frontal veins and also the veins of the upper lid and tiny vessels of the conjunctivum. A distinct thrill could be felt near the internal canthus. Almost a complete 6th nerve palsy with internal strabismus was present. Very slight pressure in the supraorbital region gave paræsthesias over ophthalmic division of 5th nerve, yet in this region no actual sensory changes were demonstrable, and the corneal reflex was present. Fundi showed dilated and tortuous retinal veins but no atrophy nor œdema. Compression of the left common carotid caused the bruit to cease, but after ten minutes of compression, numbness of right arm and leg appeared. Compression of opposite carotid, left facial or temporal arteries had no effect, yet compression of the dilated veins at the inner canthus of O. S. caused the bruit to cease almost entirely. Visual fields showed no defect and X-rays of skull were negative. Blood Wassermann and urine were negative. Blood pressure was 116/76. *Treatment:* Because of the numbness of right extremities resulting from shutting off of the left carotid, preliminary graded digital compression was employed. At first ten-minute periods of compression three times a day were used and during six weeks the periods gradually increased to one hour, three or four times a day. Numbness of opposite extremity no longer resulted. The exophthalmos and bruit, however, became worse. On May 26, 1923, under local anæsthesia, the left internal carotid was exposed and compressed with a Crile clamp for sixty minutes. During this period no numbness, anæsthesia nor paresis appeared on the opposite side, and the bruit was entirely shut off. A permanent double ligature was then made. This continued to shut off the bruit subjectively and objectively and during the next two weeks exophthalmos decreased to about one-half, the pulsation was hardly visible, and the 6th nerve palsy largely disappeared (see Fig. 9). The bruit was not audible to the patient, but after two weeks it could be very readily heard by the stethescope over the mass of dilated and pulsating veins above the inner canthus. This mass increased in size and a more distinct thrill than ever could be felt over it. On July 16, 1923, under local anæsthesia the left supraorbital and supranasal region was explored. The angular and naso-frontal veins were found to be dilated to the size of a little finger and these were doubly ligated and cut as they branched from the superior ophthalmic vein. For the following few days there was congestion about the eye and

FIG. 10.—Case II six months following ligation of dilated veins of the supraorbital region.

exophthalmos was more marked than before. Then the exophthalmos subsided and it was found that the pulsating swelling was entirely gone from the internal angle of the orbital fossa. The objective bruit, too, had disappeared, there were no signs of ocular palsies and pupil reacted normally. Some exophthalmos remained. (See Fig. 10). On January 21, 1924, the patient appeared as shown in Fig. 11. His exophthalmos had not altered during the last few months and he hears a bruit only occasionally at night while in bed. There has been, however, some increase in the size of the superficial veins of the left temporal region and of the upper lid of O. S.

CASE III.—*Accident. Resulting Pulsating Exophthalmos. Rest, Morphine, Codein and Digital Compression, Cure.* San Francisco Hospital No. 50391, a German male, aet. sixty-seven and a painter by occupation had a negative family history and past history. Present illness: On January 9, 1921, the patient was struck by a street car, was unconscious for one hour and had no subsequent memory of events occurring until eight hours after the accident. Then he noticed a severe headache. He was taken to the San Francisco Hospital. Examination showed a well developed and nourished middle-aged man in a stuporous condition. There were marked ecchymoses about each orbit and X-ray of the skull showed a left frontal fracture. No cranial nerve palsies were present. Thirteen days after the accident a paresis of the internal rectus muscle O. S. appeared and four days later there was an almost complete ophthalmoplegia externa O. S. and immobile pupil. Two weeks later, or one month after accident, a pulsating exophthalmos O. S. appeared. (See Fig. 12.) About this time subjectively as well as objectively, a blowing bruit could be heard. It was almost continuous but accentuated during systole. Exophthalmos increased and there was marked chemosis. Compression of the left common carotid shut off this bruit and sometimes it would disappear of its own accord for a time. Visual fields revealed no defect but acuity was down to 20/200 in O. S. while 20/20 in O. D. Examination of fundi showed engorgement of veins and a slightly hazy disc. Urine negative; blood-pressure not elevated. *Treatment:* Simple means such as rest, morphine, codein and digital compression of the common carotid were used. Ligation of the carotid was considered but the patient seemed to be improving so rapidly that it was not done. Two weeks after the appearance of the pulsating exophthalmos it had started to subside. In five weeks more the bruit and the ocular palsy had entirely disappeared and the exophthalmos was almost entirely gone. In another month's time there was no exophthalmos and visual acuity of O. S. was 20/70. At present no exophthalmos is present (see Fig. 12), and there are no ocular palsies. Visual acuity is O. S. now only 5/200 for a cataract has developed. The disc is somewhat pale and the arteries are much smaller than on the opposite side. Visual fields are normal. From external examination of the eyes the only abnormalities to be noted are

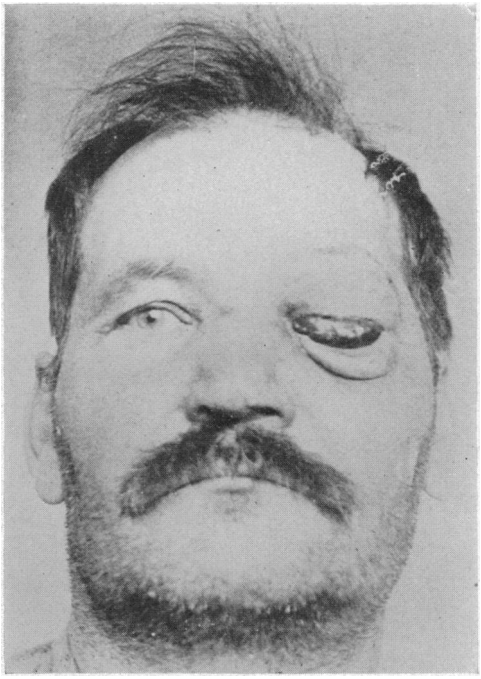

FIG. 11.—Case III one month after accident.

slight puffiness under the eye and scleral and episcleral vessels which are larger than those of the opposite globe.

CASE IV.—This case will be merely mentioned as a preliminary report* has already been made by Doctor Yoakum and the complete report will appear elsewhere.

It was a case of traumatic pulsating exophthalmos following a gunshot wound of the head. Infection of the wound followed an exploratory operation in the region of the angle of the jaw, near the entrance of the bullet. After this the pulsating exophthalmos decreased, and now, three and one-half years later, the patient is perfectly well except for a slight abducens weakness.

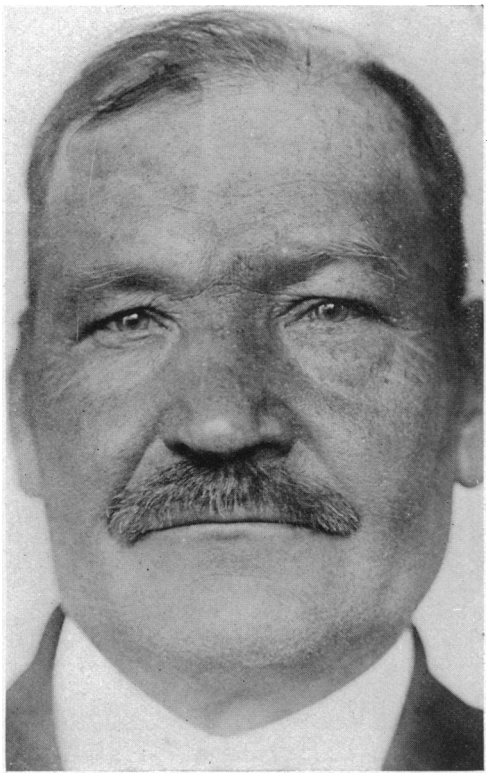

FIG. 12.—Case III two and one-half years after accident.

Diagnosis.—The clinical phenomena of pulsating exophthalmos is easily recognized yet the decision whether the underlying pathology is an arterio-venous communication, a tumor of the orbit, or an aneurism of the internal carotid or ophthalmic artery, is often difficult.

The age of the patient is of some importance in the decision, for aneurismal types are more apt to occur in middle-aged or elderly people. An aneurismal type may, however, become an arterio-venous communicating type by the rupture of a single aneurism of the internal carotid artery lying within the cavernous sinus. The traumatic cases are nearly always actual arterio-venous communication. The presence of a pulsating swelling above the inner canthus also usually means an arterio-venous type, and bruit is usually louder than with simple aneurism or with tumor. Exophthalmos is greater with tumor and the communicating type than with the aneurismal type.

With orbital tumor de Schweinitz states that the pulsating exophthalmos develops very slowly. The orbital growth, too, may often be palpable and efforts to reduce the exophthalmos are encountered with more resistance than in the other types. The bruit if present at all is very feeble.

Other conditions such as a destructive process of the roof of the orbital

* See proceeding of the Boston Society of Psychiatry and Neurology meeting at Peter Bent Brigham Hospital, January 20, 1921. Arch. Neur. and Psych., June, 1921, vol. v, p. 754.

fossa or as an orbital encephalocele may cause pulsating exophthalmos, but here no bruit is present.

Treatment.—The treatment of this condition consists of the various therapeutic procedures which tend to prevent the direct short circuiting of arterial blood into the venous system. The three very different principles that have been applied for this purpose are the following:

(1) The production of stasis or obstruction of the blood in the arteries afferent to the arterio-venous fistula, such as compression or ligation of the carotid artery.

(2) The production of stasis or obstruction to the arterial blood in the veins afferent from the arterio-venous communication, by means such as ligation or compression of superior ophthalmic vein.

(3) Methods aimed at the promotion of clot formation in both arteries and veins such as subcutaneous gelatin injections or simple rest.

The following results have been obtained by the various different methods. (See Table V.)

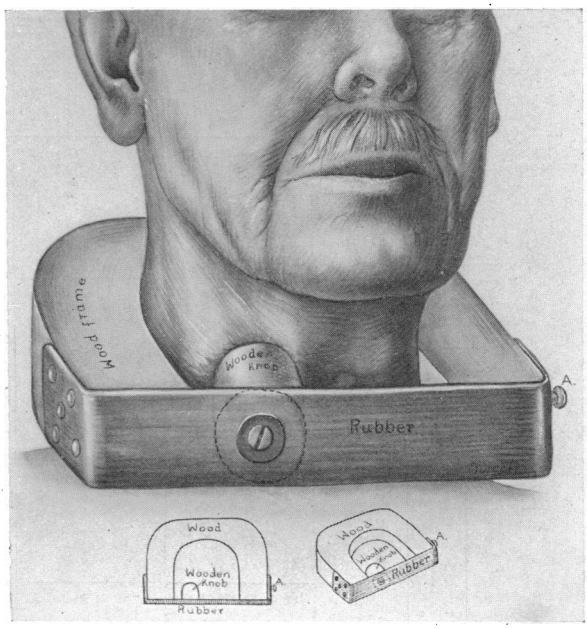

FIG. 13.—Instrument employed to compress the common carotid artery against the transverse processes of the cervical vertebræ. The wooden frame is placed in position about the neck and the rubber cross-piece is then stretched to fasten over the screw-A.

Digital Compression.—Certainly has its place as a curative therapy, as well as a preparatory procedure for carotid ligation. Since de Schweinitz and Holloway's monograph in 1907 there have been 27 cases treated in this manner with 11 patients, or 37.04 per cent. cured or improved. For the complete series of 106 cases treated by digital compression the results are less striking as there were just 26.41 per cent. cured or improved. However, by comparing the results of digital compression with those of the more radical procedures (see Table V) the reader will probably agree with the author's conclusion that this form of treatment should be thoroughly tried out as the initial therapy. It has been more effective in the treatment of spontaneous cases than those of the traumatic type.

Ligation of the common carotid was the treatment employed by Travers upon his case, the first one in the literature and this was in 1805, before the days of anæsthesia. In the literature since the publication of de Schweinitz and Holloway's monograph in 1907 there have been 84 patients

TABLE V.

Results of Treatment of Pulsating Exophthalmos.

Treatment	1923—1907			1907—1809			1923—1809	
	Series (1) Author's series	Series (2) Bedell, Rhodes, Zentmeyer, Von Nagy	Series (1) & (2) Result percentages	Series (3) De Schweinitz and Holloway	Series (4) Sattler, Reuchlin, Keller	Series (3) & (4) Result percentages	Totals	Result percentages
Digital compression	Total....23	Total.... 4		Total....11	Total... 68		Total...106	
	Cured..... 3	0	11.11	Cured.....} 3				
	Improved.... 7	0	25.93	Improved ...}	15	24.05	28	26.41
	Negative.....13	4	62.96	Negative..... 8	53	75.95	78	73.59
	Fatality..... 0	0	0.0	Fatality..... 0	0	0.0	0	0.0
	Not stated... 0	0	0.0	Not stated... 0	0	0.0	0	0.0
Ligation of common carotid	Total.....50	Total....34		Total.....34	Total...116		Total....234	
	Cured.....16	14	35.72	Cured.....} 17				
	Improved....15	12	32.14	Improved...}	80	64.60	154	65.81
	Negative.....11	7	21.43	Negative.....13	25	25.30	56	23.93
	Fatality..... 5	1	7.14	Fatality..... 4	11	10.00	21	8.98
	Not stated... 3	0	3.57	Not stated... 0	0	0.0	3	1.28
Ligation of internal carotid	Total.....25	Total.... 7		Total..... 6			Total... 38	
	Cured..... 6	1	21.87	Cured..... 1		16.66	8	21.05
	Improved....15	6	65.64	Improved.... 4		66.66	25	65.79
	Negative..... 1	0	3.12	Negative..... 1		16.66	2	5.26
	Fatality..... 3	0	9.37	Fatality..... 0		0.0	3	7.90
Bilateral ligation of carotids	Total..... 9	Total.... 2		Total..... 1	Total... 9		Total... 21	
	Cured..... 0	0	0.0	Cured.....} 0				
	Improved.... 6	1	63.64	Improved....}	6	60.00	13	61.91
	Negative..... 2	0	18.18	Negative..... 0	2	20.00	4	19.05
	Fatality..... 1	0	9.09	Fatality..... 1	1	20.00	3	14.28
	Not stated... 0	1	9.09	Not stated....0	0	0.0	1	4.76

Ligation of orbital veins alone	Total	8	Total	6		Total	3	Total	2	
	Cured	2		3	35.72	Cured	1		2	60.00
	Improved	2		1	35.72	Improved	2		0	40.00
	Negative	3		2	21.43	Negative	0		0	0.0
	Fatality	1		0	7.14	Fatality	0		0	0.0
Ligation of carotid and orbital veins	Total	10	Total	5		Total	1	Total	5	
	Cured	3		0	20.00	Cured	3		3	66.66
	Improved	1		4	33.33	Improved	1		2	33.33
	Negative	2		1	20.00	Negative	0		0	0.0
	Fatality	4		0	26.67	Fatality	0		0	0.0
Rest and medication	Total	18	Total	4		Total	6			
	Cured	2		1	13.64	Cured	1		0	16.66
	Improved	5		2	31.82	Improved	3		0	50.00
	Negative	9		1	45.45	Negative	2		0	33.33
	Fatality	1		0	4.54	Fatality	0		0	0.0
	Not stated	1		0	4.54	Not stated	0		0	0.0
Gelatin injections	Total	6	Total	7		Total	3			
	Cured	1		3	30.78	Cured	1		0	33.33
	Improved	3		2	38.46	Improved	0		0	0.0
	Negative	2		2	30.76	Negative	2		0	66.66
	Fatality	0		0	0.0	Fatality	0		0	0.0
	Not stated	0		0	0.0	Not stated	0		0	0.0

Total	19	
Cured	8	42.12
Improved	5	26.31
Negative	5	26.31
Fatality	1	5.26
Total	24	
Cured	9	37.50
Improved	8	33.33
Negative	3	12.50
Fatality	4	16.67
Total	28	
Cured	4	14.29
Improved	10	35.72
Negative	12	42.85
Fatality	1	3.57
Not stated	1	3.57
Total	16	
Cured	5	31.25
Improved	5	31.25
Negative	6	37.50
Fatality	0	0.0
Not stated	0	0.0

treated in this manner. Of these, there were 67.86 per cent. cured or improved, with a mortality of 7.14 per cent. (See Table V). The earlier series from 1809 to 1907 shows a slightly higher mortality of 10 per cent., possibly due to the septic operative wounds which occurred frequently before Lord Lister's discovery in England and Pasteur's introduction of the aseptic operating room at "Hôpital Cochin", Paris.

Ligation of the Internal Carotid.—for pulsating exophthalmos was first

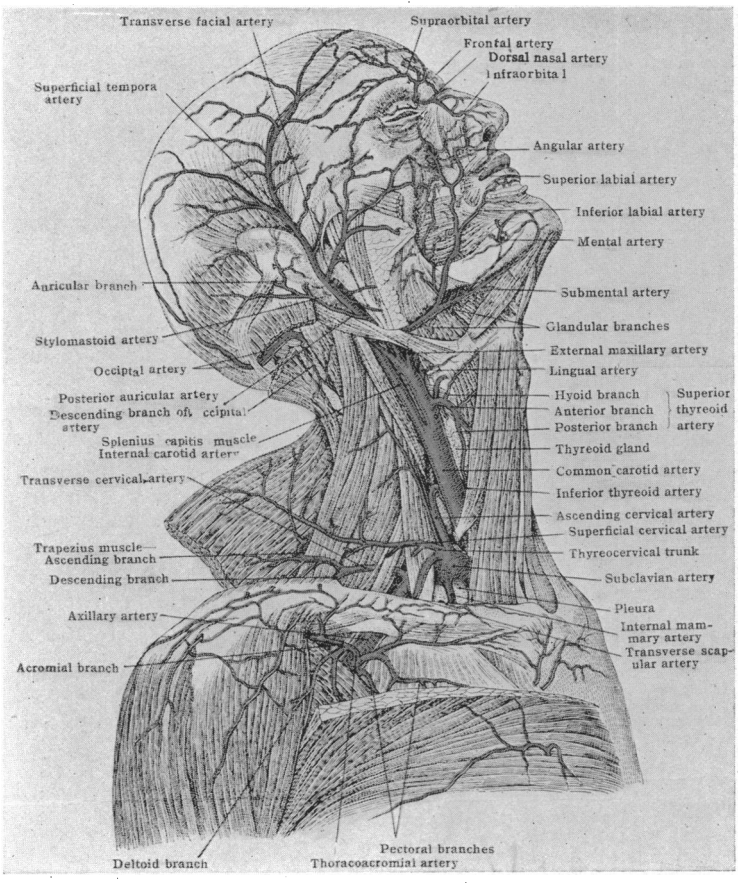

FIG. 14.—Arteries of the head and neck. (After Toldt, "Atlas of Human Anatomy," Rebman, London and New York.)

performed by Murray (New York) in 1904. Up to 1907, (de Schweinitz) there had been six cases reported with one cured, four improved, one negative, and no fatalities. The case which was cured died one month later from rupture of an aneurism of carotid near aorta. Since 1907, there have been 32 cases in which the internal carotid has been ligated with 28, or 87.51 per cent. cured or improved, and 3 per cent. or 9.37 per cent. mortality. Combining the two series there have been 38 cases in all with 86.84 per cent. cured or improved, and a 7.9 per cent. mortality. If a comparison

is to be made between these results and those of ligation of the common carotid, the common carotid series must be taken after 1904, because fatalities from infection were greater in the foregoing period.

Bilateral Ligation of the Common Carotid.—(with interval between ligations) had been performed ten times before 1907. Since then there have been 11 more cases so treated, making a total of 21, with 61.91 per cent. cured or improved, and with a 14.28 per cent. fatality. (See Table V.)

Orbital Vein Ligation.—Ligation of the dilated orbital veins at the inner angle of the orbit for pulsating exophthalmos was performed successfully by Lansdown in 1874. From that time up to the present this operation has been performed in 43 cases with 69.76 per cent. cured or improved, and 11.63 per cent. fatality. In most of the cases in which an orbital operation was performed, the patient had previously had a carotid ligation. This is unfortunate, for the merit of the orbital operation can be justly determined only from those cases in which it was the primary operation. There have been 19 of such cases with 68.42 per cent. cured or improved, and 5.26 per cent. fatality.

Rest and Medication.—Of the entire number of cases reported in the literature only very few were cured by simple means such as rest and medication. In de Schweinitz and Holloway's own series, six cases were treated in this manner and of these there was one cured, three improved and two negative. Since then there have been 23 more cases treated in this fashion, making a total of 28 cases of which there have been 4, or 14.29 per cent. cured; 10 or 35.72 per cent. improved, and 13, or 42.85 per cent. negative, and 1 or 3.57 per cent. fatality.

Gelatin Subcutaneous Injections for treatment of pulsating exophthalmos introduced by Paulesco[30] had been employed three times up to 1907, with one cure and two improvements. The total cases treated in this manner has now reached 16, of which 5 were cured, 5 improved and 6 negative. Thus 62.50 per cent. of the cases were cured or improved. A warm 2 per cent. gelatin solution has usually been used for the treatment. From 100 to 250 c.c. are injected subcutaneously every 4 to 8 days.

DISCUSSION AND CONCLUSIONS CONCERNING TREATMENT

The beneficial results from these various forms of treatment are dependent upon the decrease of blood going to or from the arterio-venous fistula, without diminishing the blood supply of that side of the brain sufficiently to cause death or hæmiplegia. In order to reduce these dangers to the very minimum, I believe that a course of carotid compression should precede every ligation operation.

In a given case of pulsating exophthalmos the treatment to be selected will largely depend upon the results of the carotid compression test. The following three examples are illustrative:

1. *If prolonged periods of carotid compression stop the bruit and do not cause signs of cerebral anæmia,* beneficial therapeutic results are to be

expected. This may be interpreted as meaning a sufficient anastomosing circulation from the opposite carotid or from the vertebral arteries to properly nourish the hemisphere, yet not a sufficient supply to maintain the arterio-venous communication. In young individuals this type of case should not have too lengthy a course of carotid compression therapy. Such treatment, if it does not early cure the condition, will merely tend to increase the anastomosing circulation so that a subsequent carotid ligation will not entirely shut off the bruit. In this type of case complete rest, subcutaneous gelatin injections and not less than one week's course of carotid compression in young individuals and three week's course in middle-aged or elderly individuals are to be recommended. In case of failure of these procedures ligation of the carotid, by the method described by the author (see below) is indicated.

2. *If the carotid compression test shuts off the bruit yet gives headache, or motor or sensory signs on the opposite side,* a thorough course of compression is indicated. Complete rest and subcutaneous gelatin injections may be carried on at the same time. The compression treatment should be given 4 to 10 times daily and increased from day to day until it is possible to constrict the artery for an hour at a time without the development of motor and sensory signs on the opposite side of the body. Figure B shows an instrument devised for the purpose of prolonged carotid compression. A thorough compression treatment is especially important in middle-aged or elderly individuals. It will prepare sufficient anastomosing circulation to alleviate the danger of subsequent carotid ligation and will, moreover, give ample time for cure in those cases in which conservative therapy is going to be successful.

3. *If the carotid compression test neither shuts off the bruit nor causes signs of brain anæmia,* then a prolonged course of carotid compression merely for therapy will not be of much value. However, a 1 to 3 weeks course, depending upon the age of the patient, will be necessary for safety as a preliminary measure to carotid ligation. The latter procedure is indicated but the surgeon will not be very confident of success.

By examination of the comparative results of common carotid and internal carotid ligation (Table V) it will be seen that there is but little advantage of one over the other. Between 1907 and 1923 there have been more cured and improved from internal carotid than from common carotid ligation, yet with this there has been a slightly greater mortality. Only as case reports increase in number will it be possible to judge which of these two procedures is the better. Either one is comparatively safe when preceded by a course of carotid compression. Out of the 13 cases in the literature treated in this manner, there were no fatalities nor ill results. It seems to the author that Murray's logic was good in suspecting fewer recurrences after internal carotid ligation. However, without a course of carotid compression it is probable that the mortality of internal carotid ligation would be greater than from common carotid ligation.

The mortality following carotid ligation may not only be reduced by the pre-operative procedure just mentioned, but also may be materially reduced by certain operative and post-operative precautions. The dangers to a patient from carotid ligation, I believe, are twofold. The first and immediate danger, that of unilateral brain anæmia from lack of blood supply, and the second, the danger from embolus or an extension of a thrombus,† which may cause an accident some days after the ligation. Both of these dangers may be minimized at the operating table by the procedure employed by the author.

Method of Carotid Ligation.—Under local anæsthesia the carotid is exposed, gently compressed and shut off with a Crile clamp. This temporary obliteration is maintained for a period of one hour. During this interval the wound is covered with gauze moistened in Ringer's solution and the patient is asked frequently whether there is headache, or feeling of numbness or weakness, etc., of the opposite extremities. Objective tests are also made. Should any signs develop, the clamp is immediately released, and the ligation abandoned until a later date after another course of digital compression. The danger of embolus and thrombosis I attempted to minimize by keeping the patient absolutely quiet for the first week after operation. He is told not to move at all, not even his arms and legs, and he is fed, turned and waited upon hand and foot by the nurses and orderlies. What is more, in the ligation itself, care is used to employ a ribbon ligature which is tied only tight enough to shut off the artery and not tight enough to injure the walls of the vessel.

Should ligation of the carotid fail and if there is a persistent enlargement and thrill of the ophthalmic veins and their branches, I believe that a ligation of these distended vessels is indicated. Even this operation although seemingly simple has a considerable mortality and with a smaller percentage of cures and improvements than carotid ligation. It should then be employed when carotid ligation has failed and under no condition should an attempt be made to do an orbital vein ligation and a carotid artery ligation at the same sitting. The results have been exceedingly unfortunate in the cases so treated. After a distal ligation of the orbital veins their lumina no doubt become filled proximally with organized clot. The extent of this process to the opening of the arterio-venous fistula is probably responsible for the cures that sometimes follow.

In case neither ligation of the internal carotid nor of the superior ophthalmic vein effects a cure, ligation of the opposite carotid may be performed. However, because of the high mortality accompanying this procedure the surgeon must assure himself by prolonged periods of digital compression that no accidents will follow. The method suggested by the author, of operating under local anæsthesia and temporarily occluding the carotid before its permanent ligation should reduce the danger of this operation.

† De Fournestraux: Les accidents cerebraux et Oculaires consecutif a la ligature de la Carotide Primitive. These, 1906-1907, No. 292.

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